```
$%^Dialog;HighlightOn=%%%;HighlightOff=%%%;

Connecting via Winsock to Dialog


Logging in to Dialog

Trying 31060000009999...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ********
ENTER PASSWORD:
 ********
Welcome to DIALOG

Dialog level 05.27.00D

Last logoff:  14oct09 15:35:49
Logon file405 03nov09 11:18:36
           *** ANNOUNCEMENTS ***
                    ***
**** October 29, 2009 - Invoices to UK customers may be delayed by
postal strike. Contact dialog.billing@dialog.com to request
email delivery, or enter HELP INVOICE for details.****

*** FREE FILE OF THE MONTH: NOVEMBER
Foodline(R): SCIENCE (File 53)

Each month Dialog offers an opportunity to try out new or
unfamiliar sources by offering $100 of free searching
(either DialUnits or connect time) in specified files.
Output and Alerts charges are not included. For more
details visit:   http://www.dialog.com/freefile/
and then take a moment to get familiar with
another great Dialog resource.

NEW FILE
***File 558, Mergent China Private Company Database
***File 457, The Lancet(R)

 >>>For the latest news about Dialog products, services, content<<<
 >>>and events, please visit What's New from Dialog at          <<<
 >>>http://www.dialog.com/whatsnew/.  You can find news about   <<<
 >>>a specific database by entering HELP NEWS <file number>.    <<<
>>>PROFILE is in a suspended state.
>>>Contact Dialog Customer Services to re-activate it.
* * *
SYSTEM:HOME
Cost is in DialUnits
Menu System II: D2 version 1.8.0 term=ASCII
                     *** DIALOG HOMEBASE(SM) Main Menu ***

 Information:
  1.  Announcements (new files, reloads, etc.)
  2.  Database, Rates, & Command Descriptions
  3.  Help in Choosing Databases for Your Topic
  4.  Customer Services (telephone assistance, training, seminars, etc.)
  5.  Product Descriptions

 Connections:
  6.  DIALOG(R) Document Delivery
  7.  Data Star(R)

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      /H = Help            /L = Logoff          /NOMENU = Command Mode


Enter an option number to view information or to connect to an online
 service.  Enter a BEGIN command plus a file number to search a database
(e.g., B1 for ERIC).
? b 411

       03nov09 11:18:54 User217743 Session D783.1
            $0.00    0.337 DialUnits FileHomeBase
     $0.00  Estimated cost FileHomeBase
     $0.08  TELNET
     $0.08  Estimated cost this search
```

```
     $0.08  Estimated total session cost   0.337 DialUnits
File 411:DIALINDEX(R)

DIALINDEX(R)
   (c) 2009 Dialog

*** DIALINDEX search results display in an abbreviated ***
*** format unless you enter the SET DETAIL ON command. ***
? s polyarteritis()nodosa and IL?6
>>>No files selected.  Use SET FILES to choose at least two files; then use
        SELECT alone to reissue this SELECT statement.
? set files biochem
   You have 29 files in your file list.
   (To see banners, use SHOW FILES command)
? s polyarteritis()nodosa and IL?6

Your SELECT statement is:
   s polyarteritis()nodosa and IL?6

          Items   File
          -----   ----
              1    72: EMBASE 1993-2009/Oct 30
              1    73: EMBASE 1974-2009/Oct 30
              1   154: MEDLINE(R) 1990-2009/Oct 30
              1   155: MEDLINE(R)_1950-2009/Oct 30

   4 files have one or more items; file list includes 29 files.

? b 155
       03nov09 11:19:47 User217743 Session D783.2
            $1.60    0.544 DialUnits File411
     $1.60  Estimated cost File411
     $0.26  TELNET
     $1.86  Estimated cost this search
     $1.94  Estimated total session cost   0.881 DialUnits

File 155:MEDLINE(R) 1950-2009/Oct 30
       (c) format only 2009 Dialog

      Set  Items  Description
      ---  -----  -----------
? s polyarteritis()nodosa and IL?6
            5932  POLYARTERITIS
            6424  NODOSA
            5702  POLYARTERITIS(W)NODOSA
             110  IL?6
      S1       1  POLYARTERITIS()NODOSA AND IL?6
? t s1/3,ab/

 1/3,AB/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

18113125   PMID: 17160656
  [Report  on the 34th meeting of the German Clinical Immunology Workgroup,
Frankfurt, 03.-04.11.2006]
  Bericht  uber  die  34.  Tagung des Arbeitskreises Klinische Immunologie,
Frankfurt, 03.-04.11.2006.
  Aries P M; Witte T; Lamprecht P
  Poliklinik  fur  Rheumatologie,  Universitatsklinikum Schleswig-Holstein,
Campus Lubeck.
  Zeitschrift fur Rheumatologie (Germany)   Feb 2007,  66  (1)  p63-4,
ISSN 0340-1855--Print   Journal Code: 0414162
  Publishing Model Print
  Document type: Congresses; English Abstract
  Languages: GERMAN
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  The  annual  meeting  of  the  Clinical  Immunology  Workgroup focused on
autoimmune  vasculitides.  The role of innate immunity, T- and B-cells, and
innovative  therapies  for  autoimmune  vasculitides was discussed. Further
topics  of the meeting were the role of endothelial microparticles, ghrelin
and  leptin,  regulatory  and  effector-memory  T-cells  in ANCA-associated
vasculitides,  as  well  as  the lethal midline granuloma, intracytoplasmic
cytokine-profile  in  Behcet's  disease,  autoantibodies  in  rheumatoid
arthritis, polyarteritis nodosa with cranial manifestation, ILT6 as genetic
marker  in  multiple  sclerosis  and  Sjogren's  syndrome,  alpha-fodrin
autoantibodies  in  multiple  sclerosis,  interferon-g  autoantibodies in a
patient  with  atypical mycobacteriosis, and autoreactive T-cells in murine
```

lupus.
? s polyarteritis()nodosa
            5932 POLYARTERITIS
            6424 NODOSA
      S2   5702 POLYARTERITIS()NODOSA
? s s2 and interleukin()"6"
            5702 S2
          183350 INTERLEUKIN
         2267701 6
           38795 INTERLEUKIN(W)6
      S3   7 S2 AND INTERLEUKIN()"6"
? T S3/3,AB/ALL

 3/3,AB/1
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

30419192   PMID: 19594951   Record Identifier: PMC2717921
  PR3-ANCA in Wegener's granulomatosis prime human mononuclear cells for enhanced activation via TLRs and NOD1/2.
  Uehara Akiko; Sato Tadasu; Iwashiro Atsushi; Yokota Sou
  Department of Microbiology and Immunology, Tohoku University Graduate School of Dentistry, Sendai, Japan. kyoro@mail.tains.tohoku.ac.jp.
  Diagnostic pathology (England) 2009, 4 p23, ISSN 1746-1596--Electronic Journal Code: 101251558
  Publishing Model Electronic
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Other Citation Owner: NLM
  Record type: In Data Review
  ABSTRACT: BACKGROUND: Anti-neutrophil cytoplasmic antibodies (ANCA) is autoantibodies characteristic of vasculitis diseases. A connection between ANCA and Wegener's granulomatosis was well established. The interaction of both ANCA phenotypes (PR3-ANCA and MPO-ANCA) with leukocytes provoked cell activation, which might be involved in the pathogenesis of ANCA-related Wegener's granulomatosis. METHODS: In this study, we examined whether PR3-ANCA sera and purified immunoglobulins from patients with Wegener's granulomatosis prime human monocytic cells for enhanced responses to microbial components in terms of production of proinflammatory cytokines. RESULTS: Flow cytometry demonstrated that stimulation with antibodies to proteinase 3 enhanced the expression of TLR2, 3, 4, 7, and 9, NOD1, and NOD2 in human mononuclear cells. The sera and purified immunoglobulins significantly primed human mononuclear cells to secrete interleukin-8 in response to microbial components via TLRs and NODs. Priming effects were also observed for the production of interleukin-6, monocyte chemoattractant protein-1, and tumor necrosis factor-alpha. On the other hand, PR3-ANCA-negative sera from patients with polyarteritis nodosa which possibly related to MPO-ANCA and aortitis syndrome as well as control sera from a healthy volunteer did not have any priming effects on PBMCs. CONCLUSION: In conclusion, PR3-ANCA prime human mononuclear cells to produce cytokines upon stimulation with various microbial components by up-regulating the TLR and NOD signaling pathway, and these mechanisms may partially participate in the inflammatory process in Wegener's granulomatosis.

 3/3,AB/2
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

16685834   PMID: 16053470
  MPO-ANCA-associated pseudovasculitis in cardiac myxoma.
  Nishio Y; Ito Y; Iguchi Y; Sato H
  Department of Neurology, Jikei University Kashiwa Hospital, Kashiwa, Japan. nishiou@mail.tains.tohoku.ac.jp
  European journal of neurology - the official journal of the European Federation of Neurological Societies (England) Aug 2005, 12 (8) p619-20, ISSN 1351-5101--Print Journal Code: 9506311
  Publishing Model Print
  Document type: Case Reports; Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: MEDLINE; Completed
  We describe a case of cardiac myxoma whose clinical presentation mimicked that of polyarteritis nodosa. The serum levels of MPO-ANCA and IL-6 were elevated on laboratory investigation and normalized after the removal of the tumor. We suggest that a 'true' vasculitic mechanism contributes to the pathogenesis of pseudovasculitis in cardiac myxoma.

3/3,AB/3
DIALOG(R)File 155:MEDLINE(R)

15683483 PMID: 14686749
Pain-related differential expression of NGF, GDNF, IL-6, and their receptors in human vasculitic neuropathies.
Yamamoto Masahiko; Ito Yasuhiro; Mitsuma Norimasa; Hattori Naoki; Sobue Gen
Department of Neurology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Showa-ku, Nagoya 466-8550.
Internal medicine (Tokyo, Japan) (Japan) Nov 2003, 42 (11) p1100-3, ISSN 0918-2918--Print Journal Code: 9204241
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
OBJECTIVE: Pain-related differential expressions of nerve growth factor (NGF), glial cell line-derived neurotrophic factor (GDNF) and interleukin-6 (IL-6), and their receptors were investigated in human vasculitic neuropathies. MATERIALS AND METHODS: The mRNA levels of pain-related neurotrophic factors, NGF, GDNF and IL-6, were examined in the sural nerves of 22 painful and non-painful patients with acute necrotizing vasculitic neuropathies, together with their concomitant soluble receptors (p75, GFR(alpha)-1 and IL-6R(alpha)). RESULTS: The mRNAs for these factors and receptors in the lesioned nerves were up-regulated to a variable extent in both groups. NGF mRNA expression was more closely correlated with that of p75 in painful neuropathy with relatively preserved large fiber density, compared with non-painful neuropathy, though the NGF mRNA level in painful neuropathy was lower than that in non-painful neuropathy. GDNF was closely associated with GFR(alpha)-1 in mRNA levels regardless of the pain state, but IL-6 was not associated with IL-6R(alpha). CONCLUSION: The differential expression of neurotrophic factors and their cognate soluble receptors in human vasculitic neuropathy suggests that NGF, which was effectively transferred to sensory axons with p75, may induce pain.

3/3,AB/4
DIALOG(R)File 155:MEDLINE(R)

14259974 PMID: 11360269
Pathology-related differential expression regulation of NGF, GDNF, CNTF, and IL-6 mRNAs in human vasculitic neuropathy.
Yamamoto M; Ito Y; Mitsuma N; Li M; Hattori N; Sobue G
Department of Neurology, Nagoya University Graduate School of Medicine, Nagoya 466-8550, Japan.
Muscle & nerve (United States) Jun 2001, 24 (6) p830-3, ISSN 0148-639X--Print Journal Code: 7803146
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The mRNA levels of nerve growth factor (NGF), glial cell line-derived neurotrophic factor (GDNF), ciliary neurotrophic factor (CNTF), and interleukin-6 (IL-6) were examined in sural nerves of 22 patients with acute necrotizing vasculitic neuropathies. NGF, GDNF, and IL-6 mRNAs were upregulated and CNTF mRNA was downregulated in the lesioned nerves, but their up- and down-regulation levels were not correlated with each other, showing that these mRNAs were independently expressed. The expression of NGF and CNTF mRNAs was clearly correlated with the degree of infiltrated macrophages and T cells, and myelinated fiber density, respectively. These findings indicate that these neurotrophic factors are differentially expressed temporally and spatially in the vasculitic nerve lesion by an underlying pathology-related process. Copyright 2001 John Wiley & Sons, Inc.

3/3,AB/5
DIALOG(R)File 155:MEDLINE(R)

10691409 PMID: 8096803 Record Identifier: PMC1554861
High circulating leukaemia inhibitory factor (LIF) in patients with giant cell arteritis: independent regulation of LIF and IL-6 under corticosteroid

therapy.
Lecron J C; Roblot P; Chevalier S; Morel F; Alderman E; Gombert J; Gascan H
URA CNRS 1172, CHRU La Miletrie, Poitiers, France.
Clinical and experimental immunology (ENGLAND) Apr 1993, 92 (1) p23-6, ISSN 0009-9104--Print Journal Code: 0057202
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Other Citation Owner: NLM
Record type: MEDLINE; Completed
Leukaemia inhibitory factor (LIF) is a cytokine which possesses a wide range of biological activities including, like IL-6, the capacity to stimulate acute phase protein (APP) synthesis. We have developed a sensitive and specific ELISA for human LIF, and tested the circulating cytokine levels in various disease states, some of which are associated with inflammation. LIF was detected in 11/20 sera from patients with giant cell arteritis (GCA), a vasculitis syndrome affecting particularly the temporal artery, characterized by panarteritis with inflammatory cell infiltration. LIF levels were considerably elevated in some patients who also displayed elevated levels of IL-6 and C-reactive protein (CRP); however, no correlation was observed between the levels of circulating LIF and levels of IL-6 or CRP. Furthermore, LIF levels were not affected by corticosteroid therapy, whereas IL-6 and CRP decreased rapidly, as clinical symptoms resolved. A putative role for LIF in the persistence of histological lesions is discussed. This is the first report of the presence of circulating LIF in sera. These results are in agreement with the complexity of induced inflammatory cytokines and corticoid regulation of APP synthesis observed in vitro and in vivo.

3/3,AB/6
DIALOG(R)File 155:MEDLINE(R)

10645127 PMID: 8432047
Elevation of cerebrospinal fluid interleukin-6 activity in patients with vasculitides and central nervous system involvement.
Hirohata S; Tanimoto K; Ito K
Department of Medicine & Physical Therapy, University of Tokyo School of Medicine, Japan.
Clinical immunology and immunopathology (UNITED STATES) Mar 1993, 66 (3) p225-9, ISSN 0090-1229--Print Journal Code: 0356637
Publishing Model Print
Document type: Case Reports; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
The pathogenesis of central nervous system (CNS) involvement in vasculitides remains unclear. We evaluated cerebrospinal fluid (CSF) interleukin-6 (IL-6) activity in relation to the CNS disease activity in vasculitides. Three patients with vasculitides of different categories who showed CNS manifestations were studied, including polyarteritis nodosa, temporal arteritis, and Behcet's disease. All three patients showed marked elevation of CSF IL-6 activity in parallel with the CNS disease activity. In one of the three patients, cerebral vasculitis was demonstrated histologically. All these patients also showed elevation of serum IL-6 activity in parallel with systemic symptoms, such as fever and/or elevation of C-reactive protein and erythrocyte sedimentation rate. These results strongly suggest that elevation of CSF IL-6 activity may underly the common pathogenetic mechanism of CNS involvement of vasculitides irrespective of their category. Taken together with the histopathological findings in one patient, the data also suggest that inflammation might not be restricted within the CNS blood vessels, but rather be extended to brain parenchyma to promote IL-6 production presumably by glial cells.

3/3,AB/7
DIALOG(R)File 155:MEDLINE(R)

10330983 PMID: 1350713
Distinct responses of interleukin-6 and other laboratory parameters to treatment in a patient with polyarteritis nodosa--a case report.
Nakahama H; Okada M; Miyazaki M; Imai N; Yokokawa T; Kubori S
Department of Medicine, Kansai Rosai Hospital, Hyogo, Japan.
Angiology (UNITED STATES) Jun 1992, 43 (6) p512-6, ISSN 0003-3197--Print Journal Code: 0203706

Publishing Model Print
Document type: Case Reports; Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The authors describe a patient in whom the serum levels of interleukin-6 (IL-6) and other laboratory parameters were monitored. The IL-6 and C-reactive protein (CRP) levels, which were extremely high before treatment, declined rapidly with administration of prednisolone. Rheumatoid factor, IgG, and platelets count declined more gradually. Thus, determination of the serum IL-6 level might be useful in diagnosing and monitoring polyarteritis nodosa.